U.S. Department of Labor

Office of Administrative Law Judges
Suite 405
2600 Mt. Ephraim Avenue
Camden, NJ 08104

(856) 757-5312
(856) 757-5403 (FAX)

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CASE NO.: 1998-BLA-01330

In the Matter of

W. JEAN WHITE, widow of
DAVID F. WHITE
Claimant

v.

CANNELTON INDUSTRIES, INC.
Employer

DIRECTOR, OFFICE OF WORKERS' COMPENSATION
Party-In-Interest

Appearances:

W. Jean White — Pro se
Paul E. Frampton, Esq. — For Employer

Before: ROBERT D. KAPLAN
Administrative Law Judge

DECISION AND ORDER
DENYING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. §§ 901, et seq., (the Act), and the regulations issued thereunder, which are found in Title 20 of the Code of Federal Regulations. Regulations referred to herein are contained in that Title.

Benefits under the Act are awarded to coal miners who are totally disabled within the meaning of the Act due to pneumoconiosis, or to the survivors of coal miners whose death was due to pneumoconiosis. Pneumoconiosis, commonly known as black lung, is a dust disease of the lungs resulting from coal dust inhalation.

On September 15, 1998, this case was referred to the Office of Administrative Law Judges for a formal hearing, and on April 20, 2000 was referred to me. The hearing was held before me in Charleston, South Carolina, on August 24, 2000, where the parties had full opportunity to present evidence and argument. The following exhibits were submitted post-hearing: reports from Drs. Ramsbottom, Levitt, and Proctor, an affidavit of David Hunt (a co-worker of the miner), Dr. Caffrey's deposition, and Dr. Fino's report. (CX 1–4; EX 5–7)[1] Based on my prior ruling at the hearing, these exhibits are now received into evidence. Claimant and Employer both filed briefs on the merits on January 16, 2001. In addition, on February 26, 2001 and March 2, 2001, Employer and Director respectively submitted briefs regarding application of the Department's amended regulations. Claimant did not file a brief regarding this matter. As my order of February 14, 2001 stated that failure to file a brief would constitute the party's position that the new regulations do not affect the outcome of the case, Claimant is in agreement with Director. Director argues that application of the amended regulations has no impact on the outcome of the case. For the reasons set forth below, I find that the amended regulations do not affect the outcome of the case. This decision is based upon an analysis of the record, the arguments of the parties, and the applicable law.

I. ISSUES

Employer has conceded that the miner had 29 years of coal mine employment with Cannelton Industries. (T 21–23) Therefore, the remaining issues presented for resolution are:

1. Whether the miner had pneumoconiosis.

2. Whether the miner's pneumoconiosis arose from his coal mine employment.

3. Whether miner's death was due to pneumoconiosis, or whether pneumoconiosis was a substantial contributor to his death.

II. FINDINGS OF FACT AND CONCLUSIONS OF LAW

A. Procedural Background

This proceeding arises from a claim for survivor's benefits filed by W. Jean White, the widow of David F. White (hereinafter "the miner"), on June 20, 1997. (DX 1) The District Director denied benefits on October 15, 1997, finding that Claimant had not shown that the miner's death was due to pneumoconiosis. (DX 24) On July 20, 1998, upon reconsideration, the District Director found that the miner had pneumoconiosis arising out of coal mine employment, and that it was a substantially contributing cause of death and factor leading to the miner's death. (DX 33) Employer requested a hearing on August 12, 1998. (DX 34)

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[1]The following abbreviations are used herein: "DX" refers to Director's Exhibit; "CX" refers to Claimant's Exhibit; "EX" refers to Employer's Exhibit; "T" refers to the transcript from the August 24, 2000 hearing.

The miner had filed an initial claim for benefits on October 28, 1977. (DX 36-1) The claim was finally denied by the Benefits Review Board on March 28, 1990. (DX 36-67)

B. Factual Background

The miner was born on February 1, 1918 and died on May 5, 1997. (DX 1) Claimant and the miner were married on April 18, 1970. (DX 5) Claimant has not remarried and she is the miner's sole dependent entitled to survivor's benefits under the Act. (DX 1)

Claimant was married to, and lived with, the miner at the time of his death. The miner began working in the mines as an underground mechanic when he was 18 years old, and he quit in 1977. (T 25–26) He began having breathing problems in the 1970s, and began using inhalers in the 1980s. Claimant described the miner as having difficulty "get[ting] the air in." (T 26–28) Claimant testified that if the miner smoked cigarettes, he did so behind her back. (T 28) The miner's previous testimony, in addition to physician records, show that he began smoking in 1938 and that he smoked approximately ½–1 pack of cigarettes a day, on and off, until 1977. (DX 36-43, pp.14; DX 10, 36-40, 36-41) Dr. Kleinerman and Dr. Fino submitted reports based on a review of the miner's medical and hospital records (some of which are found only by reference to these reports), and noted that the miner's periodic smoking continued into the 1990s. (EX 3, EX 6) I find that the miner smoked ½ pack of cigarettes a day, on and off, for at least 40 years. The miner died suddenly while walking on the beach. (T 39)

C. Entitlement

This claim was filed on June 20, 1997. Therefore, the regulations found at Part 718 are applicable. Under Part 718, benefits are provided to eligible survivors of a miner whose death was due to pneumoconiosis. § 718.205(a). Section 718.205 provides that in order to establish entitlement to survivor's benefits under Part 718, Claimant must prove that the miner had pneumoconiosis, that it arose out of his coal mine employment, and that the miner's death was due to pneumoconiosis. § 718.205. Claimant has the burden of establishing each element of entitlement by a preponderance of the evidence. Director, OWCP v. Greenwich Collieries, 512 U.S. 267 (1994).

Presence of Pneumoconiosis

There are four means of establishing the existence of pneumoconiosis, set forth at § 718.202(a)(1) through (4):

a. X-ray evidence. § 718.202(a)(1).

b. Biopsy or autopsy evidence. § 718.202(a)(2).

c. Regulatory presumptions. § 718.202(a)(3).

(1) § 718.304 – Irrebuttable presumption of total disability due to pneumoconiosis if there is evidence of complicated pneumoconiosis.

(2) § 718.305 – Where the claim was filed before January 1, 1982, there is a rebuttable presumption of total disability due to pneumoconiosis if the miner has proven fifteen (15) years of coal mine employment and there is other evidence demonstrating the existence of a totally disabling respiratory or pulmonary impairment.

(3) § 718.306 – Rebuttable presumption of entitlement applicable to cases where the miner died on or before March 1, 1978, and was employed in one or more coal mines prior to June 30, 1971.

d. Physicians' opinions based upon objective medical evidence. § 718.202(a)(4).

The U.S. Court of Appeals for the Fourth Circuit has held that, in considering whether the presence of pneumoconiosis has been established, "all relevant evidence is to be considered together rather than merely within discrete subsections of § 718.202(a)."[2] Island Creek Coal v. Compton, 211 F.3d 203, 208 (4th Cir. 2000).

X-ray evidence, § 718.202(a)(1)

Under § 718.202(a)(1) the existence of pneumoconiosis can be established by chest X-rays conducted and classified in accordance with § 718.102. The record contains the X-ray interpretations summarized in the table below.[3]

DATE OF X-RAY	DATE READ	EX. NO.	PHYSICIAN	RADIOLOGICAL CREDENTIALS	I.L.O. CLASSIFICATION
10/08/79	10/08/79	DX 36-18	Gale	BCR, B	2/1
10/08/79	01/14/80	DX 36-16	Smith	BCR, B	Unreadable
04/21/80	04/21/80	DX 36-19, 20	Gale	BCR, B	2/2
04/21/80	06/14/80	DX 36-17	Elmer	BCR, B	0/0

[2]This cases arises in the jurisdiction of the Fourth Circuit because the miner's coal mine employment took place in West Virginia.

[3]A B-reader ("B") is a physician who has demonstrated a proficiency in assessing and classifying X-ray evidence of pneumoconiosis by successful completion of an examination conducted by the United States Public Health Service. 42 C.F.R. § 37.51. A physician who is a Board-certified radiologist ("BCR") has received certification in radiology or diagnostic roentgenology by the American Board of Radiology, Inc., or the American Osteopathic Association. 20 C.F.R. § 727.206(b)(2)(iii).

DATE OF X-RAY	DATE READ	EX. NO.	PHYSICIAN	RADIOLOGICAL CREDENTIALS	I.L.O. CLASSIFICATION
01/27/81	01/27/81	DX 36-23	Bassali	BCR, B[4]	1/1
11/19/84	11/19/84	DX 36-41	Cunningham	BCR, B[5]	0/1
11/19/84	02/10/85	DX 36-41	Cole	BCR, B	0/0
11/19/84	03/28/85	DX 36-38	Renn	B	Negative
11/19/84	06/17/85	DX 36-38, 40	Wheeler	BCR, B	Negative
01/21/87	08/18/97	DX 12	Sargent	BCR, B	Negative
01/21/87	09/28/99	EX 4	Gayler	BCR, B	Negative
01/21/87	09/28/99	EX 4	Scott	BCR, B	Negative
01/21/87	09/29/99	EX 4	Wheeler	BCR, B	Negative
11/15/88	08/18/97	DX 14	Sargent	BCR, B	Negative
11/15/88	08/18/97	DX 13	Sargent	BCR, B	Negative
11/15/88	09/28/99	EX 4	Gayler	BCR, B	Negative
11/15/88	09/28/99	EX 4	Scott	BCR, B	Negative
11/15/88	09/29/99	EX 4	Wheeler	BCR, B	Negative
04/17/89	08/18/97	DX 15	Sargent	BCR, B	Negative
04/17/89	09/28/99	EX 4	Scott	BCR, B	Negative
04/17/89	09/28/99	EX 4	Gayler	BCR, B	Negative
04/17/89	09/29/99	EX 4	Wheeler	BCR, B	Negative
07/17/92	08/18/97	DX 16	Sargent	BCR, B	Negative
07/17/92	09/28/99	EX 4	Gayler	BCR, B	Negative
07/17/92	09/28/99	EX 4	Scott	BCR, B	Negative
07/17/92	09/29/99	EX 4	Wheeler	BCR, B	Negative
05/03/94	05/03/94	DX 10	Hewitt	BCR[6]	Negative
01/30/95	08/18/97	DX 17	Sargent	BCR, B	Negative
01/30/95	08/18/97	DX 18	Sargent	BCR, B	Negative

[4]www.abms.org

[5]www.abms.org

[6]www.abms.org

DATE OF X-RAY	DATE READ	EX. NO.	PHYSICIAN	RADIOLOGICAL CREDENTIALS	I.L.O. CLASSIFICATION
01/30/95	09/28/99	EX 4	Gayler	BCR, B	Negative
01/30/95	09/28/99	EX 4	Scott	BCR, B	Negative
01/30/95	09/29/99	EX 4	Wheeler	BCR, B	Negative
10/30/95	10/30/95	DX 10	Speir	BCR[7]	Negative
10/30/95	08/18/97	DX 20	Sargent	BCR, B	Negative
10/30/95	08/18/97	DX 19	Sargent	BCR, B	Negative
10/30/95	09/28/99	EX 4	Gayler	BCR, B	Negative
10/30/95	09/28/99	EX 4	Scott	BCR, B	Negative
10/30/95	09/29/99	EX 4	Wheeler	BCR, B	Negative
07/26/96	08/18/97	DX 21	Sargent	BCR, B	Negative
07/26/96	09/28/99	EX 4	Scott	BCR, B	Negative
07/26/96	09/28/99	EX 4	Gayler	BCR, B	Negative
07/26/96	09/29/99	EX 4	Wheeler	BCR, B	Negative
01/15/97	01/15/97	DX 10	Speir	BCR[8]	Negative
01/15/97	08/18/97	DX 22	Sargent	BCR, B	Negative
01/15/97	08/18/97	DX 23	Sargent	BCR, B	Negative
01/15/97	09/28/99	EX 4	Gayler	BCR, B	Negative
01/15/97	09/28/99	EX 4	Scott	BCR, B	Negative
01/15/97	09/29/99	EX 4	Wheeler	BCR, B	Negative

It is well established that the interpretation of an X-ray by a B-reader may be given additional weight by the fact finder. Sharpless v. Califano, 585 F.2d 664, 666–667 (4th Cir. 1978); Aimone v. Morrison Knudson Co., 8 BLR 1-32, 34 (1985); Martin v. Director, 6 BLR 1-535, 537 (1983). The Benefits Review Board has also held that the interpretation of an X-ray by a physician who is a B-reader as well as a Board-certified radiologist may be given more weight than that of a physician who is only a B-reader. Scheckler v. Clinchfield Coal Co., 7 BLR 1-128, 131 (1984). The X-ray evidence is overwhelmingly negative for pneumoconiosis.

Biopsy or autopsy evidence, § 718.202(a)(2)

[7]www.abms.org

[8]www.abms.org

A determination that pneumoconiosis is present may be based on a biopsy or autopsy. § 718.202(a)(2).

The record contains the report of five pathologists, Dr. Edward L. Proctor, Dr. Jerome Kleinerman, Dr. Richard Naeye, Dr. Morton Levitt, and Dr. P. Raphael Caffrey. These physicians rendered opinions based on the miner's autopsy material, and, with the exception of Dr. Proctor who performed the autopsy, also based on the miner's medical history and records. These opinions are summarized below.

Dr. Proctor (Board-certified in Anatomic and Clinical Pathology, and Forensic Pathology) performed the miner's autopsy on May 6, 1997. (DX 7, 8) The autopsy revealed that the miner's heart weighed 950 grams and that the miner had two distinct coronary artery bypass grafts. The autopsy also showed left ventricular hypertrophy and dilatation. (DX 7) Dr. Proctor reported that on gross examination he saw "prominent anthracosis" and he summarized his findings, stating that the miner's lungs showed:

> extensive anthracosis within the pleural areas. There were focal areas of intrapulmonary fibrosis with aggregates of pigment laden macrophages. These areas of fibrosis were localized to areas adjacent to bronchi and bronchial lumens. Areas of diffuse scarring were not identified. ...The lungs revealed evidence of focal fibrosis with accumulations of anthracotic pigment and numerous macrophages...consistent with coal worker's pneumoconiosis of the simple type. The simple type of coal worker's pneumoconiosis is most often characterized by areas of pigmented macrophages with surrounding fibrosis.

(DX 7) He diagnosed "arteriosclerotic cardiovascular disease" and "pulmonary congestion with anthracosis and focal fibrosis (coal workers' pneumoconiosis, simple type)." (DX 7) Dr. Proctor's autopsy report constitutes evidence of the presence of pneumoconiosis.[9] § 718.106(c).

Dr. Kleinerman (Board-certified in Anatomic and Clinical Pathology) reviewed slides of the autopsy material, and in a report dated August 1, 1999, wrote that lung tissue showed a

> small amount of black granular pigment in the subpleural and perivascular interstitial connective tissues. There is no evidence of simple CWP nor of complicated CWP. Several lung sections show localized areas of nonspecific interstitial fibrosis in the subpleural and adjacent parenchymal lung tissue. A solitary focus of hemorrhage is observed in one section. A rare lesion of centriacinar emphysema is present. There are foci of intraalveolar macrophage clusters in a rare area of lung. A small to moderate number of small arteries

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[9]In a May 20, 1998 letter, Dr. Proctor wrote that the autopsy evidence "definitely meets the criteria for simple coal workers' pneumoconiosis." (DX 32) In a September 6, 2000 letter he conducted a second review of the autopsy material and wrote that "combined with [the miner's] working history," changes in the miner's lungs were "consistent with...coal worker's pneumoconiosis." (CX 3) I find that neither of these letters add to or detract from the probative value of the initial autopsy report as it regards the presence of pneumoconiosis.

> and arterioles have a thickened fibromuscular wall. ...There are small deposits of black granular pigment in the lymph node parenchyma but no areas of fibrosis. ...The section of myocardium reveals a minimal extent of interstitial fibrosis.

(EX 3, p.6) Based on a review of the autopsy slides, the autopsy protocol, the death certificate, pulmonary function and arterial blood gas studies, X-ray reports, and medical records, Dr. Kleinerman concluded that there was no evidence of "any pulmonary disease due to coal mine dust exposure." (EX 3, p.7) Dr. Kleinerman's report is entitled to significant weight as it is well-reasoned and documented.

Dr. Naeye (Board-certified in Anatomic and Clinical Pathology) reviewed the autopsy material and submitted a report and testified in this case. (EX 1, 2) In a report of July 16, 1999, Dr. Naeye wrote that the microscopic examination of the lung tissue revealed:

> a very small amount of black pigment in the subpleural space and adjacent to some bronchioles and small arteries. All of the black deposits are far less than 1 mm in diameter and do not have any independently associated fibrous tissue. [...] Focal emphysema is absent and there are no tiny birefringent crystals associated with the anthracotic pigment. Without a history I would not have known that [the miner] had ever mined coal because some non-miners have as much black pigment in their lungs as is present in the current lungs. There are a few anthracotic macules without associated fibrosis, focal emphysema or admixed birefringent crystals. No anthracotic micronodules or larger black lesions are present. Thus no lesions are present that fulfill the minimal criteria for the diagnosis of any form of coal worker's pneumoconiosis or silicosis.

(EX 2)

Dr. Naeye also testified in a deposition on July 20, 1999. (EX 1) He reiterated the findings contained in his written report and stated that in order to diagnose pneumoconiosis, there must be "fibrous tissue mixed with the black pigment, or...a rim of destruction of lung tissue around the black pigment, which we call focal emphysema," and that, in the case of this miner, neither was present. (EX 1, p.12) He opined that the miner's fibrosis was probably residual fibrosis from a previous infection, common in older persons whose bacterial infections were not likely to have been treated with antibiotics. (EX 1, p.12–13, 17) Dr. Naeye testified that because the pigment was not associated with the fibrous tissue, the relationship between the two was "fortuitous rather than cause and effect," confirmed by the fact that the fibrosis contained no "free silica," or "birefringent crystals." (EX 1 p.11, 16–17) Finally, he disputed Dr. Proctor's opinion that the pigment was actually in macrophages and he testified as follows:

> [Dr. Proctor's] description is not correct. The anthracotic pigment is deposited in the tissues around small airways and very small arteries. It's around the second and third order respiratory bronchioles in the lungs, and its not in macrophages. The black pigment you see in macrophages is from other kinds of smoke contamination of the local environment. [The miner] retired...twenty years before he died, and there would not be any black pigment in macrophages twenty years after he left exposure to coal mine dust. Whatever was present and still around would be deposited in tissues and wouldn't be in macrophages.

(EX 1, p.14–15, 19) In addition to reviewing the autopsy material, Dr. Naeye reviewed the miner's death certificate and his medical records, which included pulmonary function studies and X-ray reports. He testified that there was no evidence of any lung disease related to coal dust exposure. (EX 1, p.15, 21) Dr. Naeye's opinion is entitled to significant weight as it is well-reasoned and documented.

Dr. Levitt (Board-certified in Anatomic and Clinical Pathology) conducted a review of the autopsy material and in a report dated September 12, 2000, Dr. Levitt found:

> [multifocal] anthracosis...mild to moderate in degree. ...[T]he fibrosis is also multifocal and tends to be perivascular, with accompanying pigmentation including anthracotic pigment in macrophages, and peribronchiolar. Sub-pleural fibrosis is mild to moderate. ...[T]here is a subtle increase in interstitial septae and multiple foci of emphysema. [...] There is evidence in my review of the slides of pulmonary fibrosis, vascular changes and focal emphysema as has been described in CWP. In addition, there are bronchitic changes that are indistinguishable from "industrial bronchitis." These changes are superimposed on changes related to a long smoking history.

(CX 2) Based on his review of medical records and the autopsy material, Dr. Levitt concluded that the miner had simple coal workers' pneumoconiosis based on his findings of pulmonary fibrosis, vascular changes, and focal emphysema. Dr. Levitt's report corroborates Dr. Proctor's autopsy findings and is entitled to substantial weight as it is well-reasoned and documented.

Dr. Caffrey (Board-certified in Anatomic and Clinical Pathology) reviewed the autopsy material and in his report dated October 5, 2000, he wrote that the autopsy material of the miner's respiratory system showed:

> evidence of acute vascular congestion noted in many areas on all these sections of lung tissue, and in some alveoli there are macrophages. In a few areas, these macrophages contains a small amount of pigment which could well be hemosiderin pigment. In these sections there is a mild amount of anthracotic pigment noted subpleurally and around small blood vessels. ...There is mild, focal interstitial fibrosis noted. There is a minimal or mild degree of centrilobular emphysema noted. The small and medium sized blood vessels show some thickening of the fibromuscular walls. No vasculitis is identified.

(EX 5) Dr. Caffrey found that the areas of the miner's lungs which showed "interstitial fibrosis" were not associated with pigment, and that the fibrosis was therefore non-specific. (EX 7, p.10) Dr. Caffrey also found mild centrilobular emphysema, but he attributed it to the miner's past history of cigarette abuse, not exposure to coal dust. (EX 6) He attributed the vascular changes and enlarged blood vessels he observed to the age of the miner, not to a pulmonary condition. (EX 7, p.15–17) Dr. Caffrey concluded that the miner had no "occupationally acquired lung disease" and that the autopsy slides showed no lung disease related to coal dust. (EX 5) Dr. Caffrey also concluded in his written report that "if a patient has only a mild degree of simple coal workers' pneumoconiosis, his pulmonary condition does not deteriorate when he leaves the mines." I find that this statement diminishes the weight of his opinion regarding the presence of pneumoconiosis, as it is irrational in light of Fourth Circuit law which recognizes the "assumption of

progressivity" of pneumoconiosis under the Act. Eastern Associated Coal Corp. v. Director, OWCP, 220 F.3d 250, 258 (4th Cir. 2000).

Discussion of Autopsy Evidence

Dr. Levitt disputed both Dr. Naeye and Dr. Kleinerman on the basis that their final determinations were inconsistent with the Pathology Standards for Coal Worker's Pneumoconiosis [hereinafter "Pathology Standards"] (published in the *Archives of Pathology and Laboratory Medicine* 103:379–432, 1979). Specifically, Dr. Levitt argued that Dr. Naeye's report of "anthracotic pigment deposited in the tissues around small airways and very small arteries" meets the Pathology Standards' criteria of pneumoconiosis, which describes that a "vascular lesion...specific for coal miners" is one in which the "small muscular artery is invested by a mantle or cuff of coal dust." (CX 2) Dr. Levitt also argues that Dr. Kleinerman's findings as set forth above, correspond to the Pathology Standards' description of a characteristic coal workers' pneumoconiosis "macular lesion" and "vascular lesion" as well.

Although there appears to be ambiguity as to whether the terms used by the pathologists describe clinical or legal pneumoconiosis in their respective reports and testimony, each of the pathologists who found no pneumoconiosis (Drs. Kleinerman, Naeye, and Caffrey) also concluded that they saw no evidence of any coal dust-induced lung disease, thus encompassing the concept of legal pneumoconiosis in their conclusions. Although there is ambiguity when Dr. Levitt refers to the Pathology Standards, because legal pneumoconiosis is encompassed within clinical pneumoconiosis, Dr. Levitt's reliance on the Pathology Standards, is nevertheless probative in terms of contradicting the reports of both Dr. Naeye and Dr. Kleinerman. Further, I note that no explicit reference to the Pathology Standards was made by either Drs. Naeye or Kleinerman. Dr. Caffrey, on the other hand, refers to the Pathology Standards and states that the lesion of coal workers' pneumoconiosis is characterized by "coal dust-laden macrophages at division of respiratory bronchials that may exist within alveoli and extend into the peribronchial, or interstitium, with associated reticulin deposits in focal emphysema," and that although he saw anthracotic pigment he found that "it did not stimulate the production of reticulin, nor did it stimulate collagen formation." (EX 7, p.7–8) When testifying about the presence of macules, Dr. Caffrey testified:

> I do not in myself, say that there is macules [sic] present. I don't know what that means March 23, 2001. I need to further define that macule. In other words, does the macule consist of anthracotic pigment with or without the production of reticulin and so forth. It's hard for me to...know what Dr. Naeye meant or how [Dr.] Levitt is interpreting him.

(EX 7, p.12–13) In addition, when asked to describe Dr. Levitt's findings of "the small muscular artery invested by a mantle or cup of coal dust" consistent with the Pathology Standards, Dr. Caffrey testified:

> the lesion of CWP...is sometimes present around these small arteries or arterials. There is thickening of the walls of these vessels. That doesn't mean that the patient has pulmonary hypertension at all. In fact, even if I were to say that [the miner] had coal workers' pneumoconiosis, to me it doesn't explain his problems...

(EX 7, p.15–16) I find that Dr. Caffrey's latter testimony does not effectively rebut Dr. Levitt's findings of vascular lesions and their relationship to pneumoconiosis, but rather attacks only Dr. Levitt's conclusion as it related to pulmonary hypertension. I find that Dr. Caffrey's testimony regarding macules does not effectively rebut Dr. Naeye's notation of "anthracotic macules."

Based on the foregoing, I find that Dr. Proctor's opinion — supported by that of Dr. Levitt — establishes that the autopsy evidence is positive for pneumoconiosis.[10] A positive finding of pneumoconiosis is further supported by the fact that Dr. Naeye himself noted the presence of "anthracotic macules." The autopsy evidence constitutes evidence of the presence of pneumoconiosis. § 718.106(c).

Regulatory presumptions, § 718.202(a)(3)

A determination of the existence of pneumoconiosis may also be made using the presumptions described in §§ 718.304, 718.305 and 718.306. Section 718.304 requires X-ray, biopsy, or equivalent evidence of complicated pneumoconiosis, a condition not present in this case. Section 718.305 is not applicable because this claim was filed after January 1, 1982. § 718.305(e). Section 718.306 is only applicable in the case of a deceased miner who died before March 1, 1978. Since none of these presumptions is applicable, the existence of pneumoconiosis has not been established under § 718.202(a)(3).

Physicians' opinions, § 718.202(a)(4)

The fourth way to establish the existence of pneumoconiosis under § 718.202 is set forth as follows in subparagraph (a)(4):

> A determination of the existence of pneumoconiosis may also be made if a physician, exercising sound medical judgment, notwithstanding a negative x-ray, finds that the miner suffers or suffered from pneumoconiosis as defined in § 718.201. Any such finding shall be based on objective medical evidence such as blood-gas studies, electrocardiograms, pulmonary function studies, physical performance tests, physical examination, and medical and work histories. Such a finding shall be supported by a reasoned medical opinion.

Section 718.201(a) (effective on January 19, 2001) defines pneumoconiosis as "a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment" and "includes both medical, or 'clinical,' pneumoconiosis and statutory, or 'legal,' pneumoconiosis." New § 718.201(a)(2) broadly defines legal pneumoconiosis as any "chronic restrictive

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[10]I give no deference to Dr. Proctor's report merely because he was the autopsy prosector, as there is no argument that performing the gross examination "provided him with an advantage over the reviewing physicians." Urgolites v. Bethenergy Mines, Inc., 17 BLR 1-20, 23 (1992); see also Bill Branch Coal Corp. v. Sparks, 213 F.3d 186, 192 (4th Cir. 2000) ("ALJs are not to credit the opinions of an autopsy prosector, to the exclusion of all other experts, solely because the autopsy prosector was the only physician to examine the whole body near the time of death.")

or obstructive pulmonary disease arising out of coal mine employment."[11] Section 718.201(b) provides:

> [A] disease "arising out of coal mine employment" includes any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.

Finally, new § 718.201(c) provides that pneumoconiosis is "recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure."[12]

As the physicians considered pulmonary function studies and arterial blood gas studies in determining the presence of pneumoconiosis, the results of these studies are summarized below.

The record contains the following pulmonary function studies:[13]

DATE	EX. NO.	PHYSICIAN	AGE	FEV_1	FVC	MVV	FEV_1/ FVC	EFFORT	QUALIFIES
10/08/79	DX 36-14	Cardona	60	3.50	—	115	—	Good	No
01/27/81	DX 36-23	Rasmussen	62	3.61	4.44	152	81%	—	No
11/19/84	DX 36-36	Abernathy	66	3.62	4.21	129	86%	Good	No
10/21/91	DX 11	Trask	73	3.08	3.61	—	85%	—	No

[11]Pursuant to the Preliminary Injunction Order of U.S. District Judge Emmet G. Sullivan on February 9, 2001 (staying action on pending Black Lung cases except where the validity of the amended regulation is not at issue), in an order of February 14, 2001, the undersigned directed the parties to submit briefs addressing whether the application of amended regulatory provisions at 20 C.F.R. §§ 718.104(d), 718.201(a)(2), 718.201(c), 718.204(a), 718.205(c)(5), or 718.205(d) affect the outcome of the instant claim. In regards to the amended regulation's definition of legal pneumoconiosis, Director contends that this is consistent with, and codifies, existing Fourth Circuit law which recognizes this principle. Gulf and Western Industries v. Ling, 176 F.3d 226, 231–32 (4th Cir. 1999); Richardson v. Director, OWCP, 94 F.3d 164, 166 n.2 (4th Cir. 1996). (Director's Brief at 3) I agree and further find that there are no physician opinions of record that hinge on a dispute as to whether the miner had a restrictive or obstructive condition, and therefore had, or did not have, pneumoconiosis. Thus, amended regulation § 718.201(a) does not affect the outcome of this case.

[12]Director contends that this is consistent with, and codifies, existing Fourth Circuit law which recognizes this principle. Eastern Associated Coal Corp. v. Director, OWCP, 220 F.3d 250, 258 (4th Cir. 2000). Director argues, therefore, that application of the amended regulation has no impact on the outcome of the case. (Directors's Brief at 4–5). I agree.

[13]None of the pulmonary function studies were conducted after January 19, 2001 and therefore the provisions found at amended § 718.103 do not apply to these studies. § 718.101.

DATE	EX. NO.	PHYSICIAN	AGE	FEV_1	FVC	MVV	FEV_1/ FVC	EFFORT	QUALIFIES
12/04/92	DX 10	Cohen	74	2.60	3.16	—	82%	—	No
02/09/95	DX 11	Trask	76	2.90	3.49	77	83%	—	No

The record contains the following arterial blood gas study:

DATE	EX. NO.	PHYSICIAN	pCO_2	pO_2	QUALIFIES
01/27/81	DX 36-23	Rasmussen	39 33*	68 87*	No No*

* post-exercise

Dr. Ramsbottom and Dr. Fino submitted current opinions regarding the presence of pneumoconiosis. Dr. Trask's medical records are also in evidence, as is a previous opinion of Dr. Abernathy. This evidence is summarized below.

Dr. John G. Ramsbottom, Sr. (miner's treating physician/cardiologist)[14] wrote a letter on September 1, 2000 and reported that the miner:

> had a history of black lung disease as well as heart disease. Over the course of ten years that I took care of him, he continually complained of a lot of shortness of breath with general symptoms indicative of numoconiosis [sic] as well as chronic obstructive pulmonary disease and heart disease. [The miner] repeatedly complained of labored breathing and inability to catch his breath and in my medical opinion, pneumoconiosis had a lot to do with his demise and general medical condition.

(CX 1) Dr. Ramsbottom's records, which are also part of the record, contain reports and letters he received from other physicians.[15] (DX 10) This material contains no diagnosis of pneumoconiosis nor any other pulmonary condition. In fact, in a letter from Dr. Cohen to Dr. Ramsbottom, dated December, 4, 1992, Dr. Cohen wrote that "[i]t is difficult to say what is going on with [the miner]. Either he has some sort of bronchospastic disorder which is not evident at the time of his evaluation, or the etiology of his dyspnea is non-pulmonary." (DX 10) In a letter of July 26, 1996 to Dr. Ramsbottom, Dr. Trask wrote about the miner's symptoms of coughing and sputum production and concluded: "As always, I have had

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[14]Employer contends that amended § 718.104(d) affects the outcome of the case because it changes the manner in which a treating physician's opinion is weighed. (Employer's Brief at 2) As Director argues, however, Employer overlooks that § 718.101 provides that revised § 718.204 applies only to evidence adduced after January 19, 2001, and therefore does not apply to the instant case. (Director's Brief at 2). I agree.

[15]Dr. Ramsbottom's handwritten notes, which are part of the record, are illegible.

a difficult time discerning what is due to heart failure and what is due to [the miner's] lungs." Dr. Trask did not diagnose pneumoconiosis in this letter to Dr. Ramsbottom. I find that Dr. Ramsbottom's September 1, 2000 letter is not supported by any objective data, and does not support a finding of pneumoconiosis.

Dr. Joseph Trask (Board-certified in Internal Medicine and Cardiovascular Disease; miner's treating physician) submitted pulmonary function studies which appear to be related to his previous treatment of the miner. These pulmonary function studies contain no diagnosis of pneumoconiosis or other pulmonary condition related to coal dust exposure. As discussed above, in a letter of July 26, 1996 to Dr. Ramsbottom, Dr. Trask wrote about the miner's symptoms of coughing and sputum production and concluded: "As always, I have had a difficult time discerning what is due to heart failure and what is due to [the miner's] lungs." I find that this is equivocal and that none of these reports support a finding of pneumoconiosis.

Dr. Abernathy (qualifications not of record) noted in a report dated November 21, 1984, that the miner's symptoms of shortness of breath, coughing, sputum production, and dark mucous. He performed an examination and laboratory tests. He reported that a chest X-ray revealed fibrosis. He diagnosed pneumoconiosis despite "normal pulmonary mechanics," but determined that the miner was not totally disabled. I find that his report is supported by objective data and, despite its age, supports a finding of pneumoconiosis. (DX 36-36)

Dr. Gregory Fino (Board-certified in Internal Medicine and Pulmonary Disease) reviewed the medical evidence of record. He reviewed X-rays, arterial blood gas and pulmonary function studies, and the autopsy material. He wrote that "as to the presence or absence of pneumoconiosis, I personally would clearly rely on Drs. Naeye and Kleinerman. Both of them helped to write the standards for the pathologic detection of pneumoconiosis." He further wrote that there is "insufficient objective medical data to justify a diagnosis of simple coal workers' pneumoconiosis." I find that Dr. Fino's report is not well-reasoned because he bases his conclusion primarily on the findings of Dr. Naeye and Dr. Kleinerman. As I have found that these pathology reports were outweighed by the reports of Dr. Proctor and Dr. Levitt, I find that this undermines the weight to be attributed to Dr. Fino's opinion regarding the presence of pneumoconiosis.

Weighing the Medical Evidence Together

Despite the negative X-ray reports, in light of the highly probative autopsy evidence, I find that the opinions of Dr. Proctor and Dr. Levitt, in combination with the opinion of Dr. Abernathy, establish that the miner had pneumoconiosis.

Pneumoconiosis Arising Out of Coal Mine Employment

Because the miner worked for over 10 years in coal mine employment, the presumption of causality provided for in 20 C.F.R. §§ 718.203(b); 718.302 is applicable. The record does not suggest any other employment that could be the cause of the miner's pneumoconiosis, nor does any evidence rebut this presumption. Therefore, Claimant is entitled to a finding that if he had pneumoconiosis, that it arose out of coal mine employment. 20 C.F.R. §§ 718.203(b); 718.302.

Death Due to Pneumoconiosis

As the survivor's claim was filed after January 1, 1982, under § 718.1 Claimant next must show that the miner's death was due to pneumoconiosis. Death due to pneumoconiosis may be established under § 718.205 (effective on January 19, 2001) by any of the following criteria:

1. Competent medical evidence establishes that pneumoconiosis was the cause of the miner's death.

2. Evidence that pneumoconiosis was a substantially contributing cause or factor leading to the miner's death, or that death was caused by complications of pneumoconiosis.

3. Under 718.304, the miner suffered from a chronic dust disease of the lung and chest X-ray evidence shows one or more large opacities (greater than 1 centimeter), biopsy or autopsy shows massive lesions in the lung, or the other evidence (in accord with acceptable medical procedures) show a condition which could reasonably be expected to yield such large opacities or massive lesions.

4. Evidence that pneumoconiosis hastened the miner's death.

§ 718.205(c)(1)–(5); Shuff v. Cedar Coal Co., 967 F.2d 977 (4th Cir. 1992).[16]

Eight physicians, Drs. Trask, Proctor, Kleinerman, Naeye, Levitt, Ramsbottom, Caffrey, and Fino, rendered opinions regarding the cause of the miner's death.

Dr. Trask signed the miner's death certificate and wrote that the immediate cause of the miner's death was "ventricular fibrillation." He reported that the underlying causes leading to the immediate cause of death were "ischemic cardiomyopathy" and "coronary artery disease." (DX 6) Dr. Trask submitted no additional report or opinion regarding the cause of death. Dr. Trask's opinion, as reported in the death certificate, does not support a finding that pneumoconiosis contributed to the miner's death.

In his initial autopsy report, Dr. Proctor wrote that the cause of death was arteriosclerotic cardiovascular disease, and that "the cause of death [was] natural." He listed "pulmonary congestion with focal fibrosis and anthracosis (coal workers' pneumoconiosis) under the category of "significant findings." (DX 7) He did not state a clear opinion that pneumoconiosis contributed to the miner's death. In a letter of December 12, 1997, Dr. Proctor wrote that he had reviewed the autopsy findings and concluded that the miner's pneumoconiosis "contributed to the exacerbation and progression of [the miner's] cardiac problems." (DX 31) He explained that although pneumoconiosis did not cause the miner's cardiac problems, the miner's pulmonary condition caused the "heart to work much harder to adequately profuse

[16]Director contends that application of this amended regulation does not affect the outcome of the case because the Fourth Circuit Court of Appeals has held that pneumoconiosis constitutes a "substantially contributing cause" where it shortens life or hastens death, even "briefly." § 718.205(c)(1)–(5); Shuff v. Cedar Coal Co., 967 F.2d 977 (4th Cir. 1992). I agree.

the system with oxygenated blood." (DX 31) It appears that Dr. Proctor only reviewed the autopsy material again, and did not review any medical records of the miner. In addition, in neither his autopsy report, nor in his 1997 letter, does he record any information regarding the miner's history of cigarette smoking and its impact on the miner's pulmonary condition. In light of that, and given that this opinion is inconsistent with his initial findings (where he noted pneumoconiosis only as a "significant finding"), I find that his opinion on cause of death is entitled to minimal weight.

Dr. Levitt wrote that there was "no doubt that the miner had significant pulmonary disease that was caused by the cigarette smoking," but he concurred with Dr. Proctor that the simple coal workers' pneumoconiosis "contribute[d] to the exacerbation of [the miner's] cardiac symptoms." (CX 2) After describing the "deterioration of [the miner's] pulmonary function" based on his review of the miner's pulmonary function and arterial blood gas studies, he wrote:

> [S]eparating ischemic...heart disease due to atherosclerosis, from small vessel...heart disease due to a variety of insults such as smoking can be difficult. Thus, it is traditional to look for "characteristic CWP lesions," or other evidence of coal-dust related lung disease, such as silicosis, to determine the presence or absence of lung disease. [...]There is no question in my mind...that [the miner] had significant pulmonary disease that contributed to his death, although the principal cause of death was cardiac. ...[T]he presence of coal-dust related diseases potentiate the damage due to smoking in a super-additive fashion. Finally, there are pulmonary arterial changes of pulmonary hypertension, which further support the presence of significant pulmonary disease, although in and of themselves do not prove or disprove it. I am unable to reach a conclusion about the presence or absence of so-called "cor pulmonale (right heart failure due to advanced pulmonary disease)," but the weight and size of the heart...and the presence of pulmonary hypertension is suggestive.

(CX 2) I find that Dr. Levitt's opinions regarding pulmonary hypertension and cor pulmonale are equivocal and inconclusive. Nevertheless, I infer that Dr. Levitt is of the opinion that pneumoconiosis exacerbated the miner's cardiac symptoms because of a deterioration in pulmonary function, as he wrote "[t]aken superficially, [the results of the early studies] indicate absence of clinically significant obstructive or restrictive lung disease..., but the question of deterioration in [the miner's] pulmonary function is clearly evident in the PFTs from the period more proximal to the [miner's] death. (CX 2) I find that Dr. Levitt's opinion is well-documented and entitled to significant weight.

Dr. Kleinerman opined that even if the miner had pneumoconiosis, it did not contribute to the miner's death. (EX 3) He attributed death to ventricular fibrillation, ischemic cardiomyopathy and atherosclerotic coronary arterial disease. He disagreed with Dr. Proctor that pneumoconiosis contributed to the miner's cardiac disease, on the basis that the autopsy material was negative. He found that the miner's pulmonary function studies were normal and showed no evidence of an obstructive or restrictive lung dysfunction. Moreover, he found that the miner's arterial blood gas studies were normal. As the studies had been conducted as early as 1979, and as late as 1995, Dr. Kleinerman surmised that the miner's pulmonary function was normal for many years. He concluded therefore, that the pneumoconiosis did not contribute in any way to the miner's cardiac dysfunction, nor cause, contribute, nor hasten the miner's death. (EX 3) Because Dr. Kleinerman's ultimate opinion (i.e., that pneumoconiosis did not hasten

death) is not premised solely on his erroneous finding of no pneumoconiosis, and rather is grounded on several other rational factors, I find that his opinion is well-documented and reasoned, and entitled to significant weight.

Dr. Naeye opined that death was due to "coronary artery disease" and "damage in the microcirculation as a result of cigarette smoking." (EX 2, p.22) He wrote that death was due to

> complications of coronary artery disease and likely damage in the microcirculation of his heart that were a result of his cigarette smoking. Being absent CWP did not cause any impairments in the lung function or hasten this man's death.

(EX 1) I find that Dr. Naeye is of the opinion that pneumoconiosis could not hasten the man's death because it was absent, whereas I have found that the miner had pneumoconiosis. For this reason, I find that his opinion is entitled to no weight.

Dr. Caffrey found that the miner's death was not due to pneumoconiosis, and he wrote that even if the miner had pulmonary problems, those problems were as a result of smoking cigarettes, and that the miner's death was due to his cardiac problems. Dr. Caffrey wrote that the miner's "heart was failing rapidly" and that he "was being treated for fibrillation, congestive heart failure, etc." (EX 5) Furthermore, he testified that "simple CWP that is not high category does not cause pulmonary complications." (EX 7, p.19) In fact, when asked again "if on autopsy, the pathology demonstrates that the pneumoconiosis that is present is minimal, then it's not going to contribute to pulmonary impairment or death?," Dr. Caffrey answered: "Yes." (EX 7, p.19) I find that this statement is not rational under the Act, which provides that simple pneumoconiosis can be totally disabling and can contribute to death. I therefore find that Dr. Caffrey's opinion is not entitled to any weight.

Dr. Fino wrote that had the miner established that he had pneumoconiosis, the medical evidence established that his death was nevertheless due to cardiac disease, and that his cardiac disease was not caused by coal dust. He specifically rebutted Dr. Proctor's opinion (and ultimately the opinion of Dr. Levitt who agreed with Dr. Proctor) that the miner's pneumoconiosis contributed to the exacerbation and progression of the miner's cardiac problems. (EX 6) He noted that even if the miner had pneumoconiosis,

> there was nothing to suggest a pulmonary problem that could conceivably contribute to his heart disease. He had no chronic resting hypoxia, and all of his lung function studies were normal. There was an episode of hypoxia (with a p02 of 60) during January of 1995, but that was due to heart failure. [...]Regardless of whether or not pneumoconiosis was present, there was absolutely no evidence of any respiratory impairment or pulmonary disability. ...Regardless of whether or not pneumoconiosis was present, this man died due to cardiac disease. That disease was not caused, contributed to, or hastened by the inhalation of coal mine dust.

(EX 6) Although Dr. Fino did not find pneumoconiosis, he went on to consider the effect of presumed pneumoconiosis. I find that his conclusion that the presumed pneumoconiosis did not hasten death is entitled to significant weight as it is well-documented.

Dr. Ramsbottom stated in his September 1, 2000 letter that "[the miner] repeatedly complained of labored breathing with inability to catch his breath and in my medical opinion, ~~numo~~pneumoconiosis [sic] had a lot to do with the miner's demise and general medical condition." (CX 1) This letter is not supported by any objective data, except the reference to the miner's subjective complaints. I find that this report is entitled to minimal, if any, weight.

For the reasons set forth above, I find that the reports of Dr. Levitt and Dr. Proctor (finding that pneumoconiosis contributed to death) are outweighed by the reports of Dr. Kleinerman and Dr. Fino (finding the opposite).[17] The latter represent the opinions of both a qualified pathologist and pulmonologist, who both reviewed the pulmonary function studies and characterized the miner's lung function as "normal." Only Dr. Levitt, and not Dr. Proctor, reviewed these studies when arriving at his contrary conclusion. I find that the evidence put forth by the Claimant is insufficient to establish that the miner's death was due to pneumoconiosis.

D. Conclusion

I find that although the miner had pneumoconiosis, there is insufficient proof that the pneumoconiosis hastened or contributed to his death. Based on the foregoing, I find that the Claimant has not proven entitlement to benefits pursuant to § 718.205(c).

ORDER

The claim of W. JEAN WHITE for benefits under the Act is DENIED.

A
Robert D. Kaplan
Administrative Law Judge

Camden, New Jersey

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefit Review Board within 30 (thirty) days from the date of this

[17] Although Claimant submitted an affidavit of David Hunt, the miner's co-worker, a layperson's opinion with regards to the issue of cause of death is not probative here. § 718.205(c). Hunt's opinion that pneumoconiosis "caused or exasperated [the miner's] other problems, thereby making 'black lung disease' the initial and primary contributing factor that caused his death," is contradicted by the record. Mancia v. Director, OWCP, 130 F.3d 579, 588 (3d Cir. 1997) (permitting an ALJ to consider "uncontradicted relevant lay testimony where it corroborates the medical testimony of a treating physician and is consistent with the medical records.")

Decision by filing a Notice of Appeal with the Benefits Review Board at P.O. Box 37601, Washington, D.C. 20013-7601. A copy of this Notice of Appeal must also be served on Donald S. Shire, Associate Solicitor for Black Lung Benefits, 200 Constitution Avenue, N.W., Room N-2117, Washington, D.C., 20210.